In one place, the authors mentioned that 17 cases received hormonal treatment (HT), while in the next sentence the number of cases receiving HT reached 31 cases. It seems reasonable that the patients were advised to withdraw from using any hormonal substance, and that is the third prerequisite for randomization. In addition, the use of estrogenic compounds should cease before any elective surgery in order to avoid thromboembolic accidents. More usefully, they could have added up all cases that were on HT after surgery and compared their symptoms with the other 21 be ceased that were not. Such a comparison could more accurately specify the role of HT on changes in the symptoms.

If randomization had been done correctly, we would have expected that the number of suspended and unsuspended ovaries (which were 20 and 27 ovaries, respectively), and suspended ovaries on each side (which are not mentioned in this study) would be equal.

The authors stated that the comparable rate of post-operative adhesions examined by transvaginal ultrasound in their study with previous studies which used laparoscopy indicated that their findings are reassuring. Such judgment would be true if they had used both diagnostic procedures (transvaginal ultrasound and laparoscopy) on the same cases or on a similar study population. Comparable percentages of cases with adhesions using both diagnostic procedures prove nothing. Furthermore, if we conducted a diagnostic study on the same cases and considered laparoscopy as the gold standard, the percentages of cases with false-negative and false-positive adhesions by vaginal ultrasound may be nearly equal and this could result in a comparable percentage of adhesions by the two methods. Alternatively, maybe the cases with adhesions based on laparoscopy are healthy according to a transvaginal ultrasound and vice versa.

**References**


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**Reply: Criticizing the effect of ovarian suspension on adhesions in laparoscopic surgery for endometriosis**

Sir,

We thank Mehdizadehkashi and colleagues for their interest in our study (Hoo et al., 2014). Our study was a prospective, within-group comparison, double-blind randomized control trial. This implies that comparisons were made within and not between individuals. We stated in the discussion that we decided to use this type of trial as it is considered methodologically sound and free of disadvantages that may affect the quality of parallel group trials.

We stated very clearly in our paper that the inclusion criteria were the presence of severe bilateral disease (as scored using the revised AFS staging) requiring extensive dissection of both pelvic side walls and/or rectovaginal space with preservation of the ovaries and the uterus. As severe endometriosis often occurs without endometriomas being present in the ovaries, the presence of endometrioma was not a part of the inclusion criteria. We did perform a secondary analysis, however, which showed that ovarian cystectomy did not have a significant effect on the ipsilateral adhesion rates.

With regard to hormonal treatment we made it clear that 14 women had hormonal treatment pre-operatively and an additional 17 women were treated with hormones post-operatively. Statistical comparisons were made between 31 women who had hormonal treatment (either pre- or post-operatively) and 21 women who did not receive any hormonal treatment. We found no significant difference in the adhesion rates between the two groups. We also made it clear, however, that our study was not powered to evaluate the effect of post-operative hormonal treatment on the presence of adhesions.

The number of suspended and unsuspended ovaries was the same, i.e. 52. There was evidence of ovarian adhesions in 20/52 suspended ovaries compared with 27/52 in unsuspended ovaries. These numbers therefore refer to the primary findings in our study and not to the number of ovaries randomized to intervention and non-intervention.

We accept that a second-look laparoscopy is still perceived by many as a gold standard to assess for the presence of pelvic adhesions. However, little is known about the reproducibility of laparoscopic diagnosis of pelvic adhesions, which is likely to be as operator dependent as in non-invasive diagnostic methods. Second-look laparoscopy is costly, it is associated with surgical risks and many asymptomatic and minimally symptomatic women are reluctant to undergo further surgery which confers no obvious benefit to them. As mentioned in the discussion, recent improvements in the quality of ultrasound equipment and the examination technique showed that transvaginal ultrasound examination is an accurate and reproducible test to diagnose pelvic adhesions and to assess their severity (Holland et al., 2010; Hudelist et al., 2011; Holland et al., 2013).

**References**


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**GnRH agonist triggering in high-risk patients**

Sir,

We were interested in the article published in one of the recent issues of Human Reproduction, entitled ‘Consistent high clinical pregnancy rates and low ovarian hyperstimulation syndrome rates in high-risk patients after GnRH agonist triggering and modified luteal support: a retrospective multicentre study’ written by Iliodromiti et al. (2013). Here, we would like to discuss some important points concerning the article.

Iliodromiti et al. (2013) in their three-centre retrospective analysis included 275 patients, at high risk of developing ovarian hyperstimulation syndrome (OHSS), who received a GnRH agonist trigger followed by a bolus of 1500 IU hCG 1 h after oocyte retrieval. GnRH agonist trigger followed by a modified luteal phase support with one bolus of 1500 IU hCG was developed by Humaidan et al. (2005, 2010). As stated in the article, within an hour of oocyte retrieval, centres one and three (UK, Australia) administered 1500 IU recombinant hCG (Merck Serono) for luteal support. There is only one form of recombinant hCG which consists of 250 microgram /0.5 ml choriogonadotropin alfa and equals to 6500 IU. We would like to ask how these centres administered 1500 IU recombinant hCG?

The second point we question is the discordance between baseline characteristics and ovarian response of the study population. The study population was presented as having increased risk of an excessive ovarian response, but the total number of patients with polycystic ovary syndrome was 16.7%. Total FSH, peak estradiol levels, and the number of oocytes collected do not represent high-risk patients. In our opinion, the 0.72% incidence of severe OHSS ratio represents for moderate-risk patients.

**References**


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**Reply: GnRH agonist triggering in high-risk patients**

Sir

We are grateful for the interest shown by Gurbuz and colleagues in our manuscript reporting our multicentre experience of modified luteal support after GnRH agonist trigger (Iliodromiti et al., 2013). Rather than as reported in both the Australian and Belgian centre 1500 IU urinary hCG (Pregnyl) was used. In the UK centre, as 1500 IU urinary hCG was not available, this was approximated by using a fractional dose of the Ovitrelle prefilled pen (Merck Serono). Specifically 25% of the total number of clicks to deliver the full dose was dialled prior to injection (8/32) and a dose approximating 1625 IU hCG was administered.

We acknowledge that there was discordance between baseline characteristics (antral follicle count and anti-Müllerian hormone (AMH)) that provide an estimate of potential ovarian response and the actual ovarian response. We would however respectfully suggest that the observed ovarian response does not reflect the inherent risk profile of the patients, but rather successful modification of the stimulation strategy. We have previously shown the utility of antral follicle count and AMH in predicting the range of ovarian responses (Nelson et al., 2007; Bredkaier et al., 2013a,b) and that the chosen stimulation strategy of a GnRH agonist with a low dose of exogenous FSH can modify oocyte yield, particularly in potential high responders (Macklon et al., 2006; Nelson et al., 2009). The ability to use ovarian biomarkers to identify high-risk patients, stratify their treatment to less aggressive stimulation strategies, modify their response and associated risk while still maintaining excellent...